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CASE REPORTS

Out-of-Hospital Automatic Cardioversion of Ventricular Tachycardia

RODOLPHE RUFFY, MD, FACC, PRISCILLA SMITH, RN, MICHAEL LASETER, MD, ROOP LAL, MD, SUNG SOON KIM, MD, FACC

St. Louis, Missouri

A 42 year old man who survived sudden cardiac death was treated with an automatic implantable cardioverter/defibrillator. After a 5 month symptom-free interval, the patient received two internal discharges in the conscious state while wearing an ambulatory electrocardiographic recorder. Analysis of the tape revealed that both discharges were activated by two bursts of polymorphous ventricular tachycardia, the first one occurring at the end and the second at the onset of episodes of slow, hemodynamically stable monomorphous ventricular tachycardia. This case illustrates the reliability of the automatic implantable cardioverter/defibrillator as an antiventricular tachycardia device and the problem posed by its exposure to nonsustained ventricular tachycardia.

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The automatic implantable cardioverter/defibrillator (AICD, Intec Systems, Inc.) is an effective form of treatment for patients at risk of recurrent out-of-hospital cardiac arrest or other forms of hemodynamically devastating ventricular tachyarrhythmias (1,2). This report illustrates the reliability of this instrument as an antiventricular tachycardia device and provides, to the best of our knowledge, the first electrocardiographically documented illustration of successful out-of-hospital treatment of ventricular tachycardia by automatic cardioversion. It also documents a less desirable by-product of the device, namely, the occurrence of automatic, internal discharges when symptoms of paroxysmal arrhythmia are minimal or nonexistent.

Case Report

The patient, a 42 year old white man, had a large anteroseptal myocardial infarction with subsequent aneurysm formation in 1978. He was referred to this hospital in 1980 for management of recurrent sustained ventricular tachycardia that was refractory to multiple drug trials. He was then referred elsewhere for surgical treatment guided by intraoperative mapping, but ventricular tachycardia remained inducible by programmed stimulation postoperatively. He was placed on a regimen of quinidine and phenytoin and suffered no recurrence until December 1983 when he had two episodes of ventricular tachycardia, each requiring cardioversion. His plasma quinidine level was found to be low and the daily dose of the drug was increased.

Cardioverter/defibrillator implantation. In April 1984, he sustained an episode of out-of-hospital cardiac arrest, requiring prolonged cardiopulmonary resuscitation and multiple direct-current shocks, from which he recovered well. Subsequently, the patient underwent programmed ventricular electrical stimulation in a drug-free state; this induced sustained ventricular tachycardia at a rate of 200 beats/min. Antiarrhythmic therapy with amiodarone and lorcainide was instituted and on April 21, 1984 a cardioverter/defibrillator was implanted and set to intervene at a rate of tachycardia above 176 beats/min. After the patient's recovery from the thoracotomy and 18 days after initiation of combined antiarrhythmic therapy, electrophysiologic testing was repeated and demonstrated persistent inducibility of sustained ventricular tachycardia.

Two varieties of ventricular tachycardia were induced by double extrastimulation of the right ventricular apex: The first had a rate of 140 beats/min, was monomorphous and produced no apparent hemodynamic alteration; it was not terminated by the automatic device, but by a single programmed extrastimulus. The second variety had an initial rate of 225 beats/min, was polymorphous, rapidly degenerated into ventricular fibrillation and was terminated by a single automatic shock delivered after a charge-up time of 8.6 seconds. The patient was discharged from the hospital and his progress was followed up on an outpatient basis.

Outpatient follow-up. The patient was soon able to resume his usual activities and remained symptom-free until

From The Arrhythmia Service, Cardiology Division, Jewish Hospital at Washington University, St. Louis, Missouri. Manuscript received December 26, 1984; revised manuscript received March 4, 1985, accepted March 15, 1985.

Address for reprints: Rodolphe Ruffy, MD, Director, Arrhythmia Service, Jewish Hospital at Washington University, 216 South Kingshighway, St. Louis, Missouri 63110.

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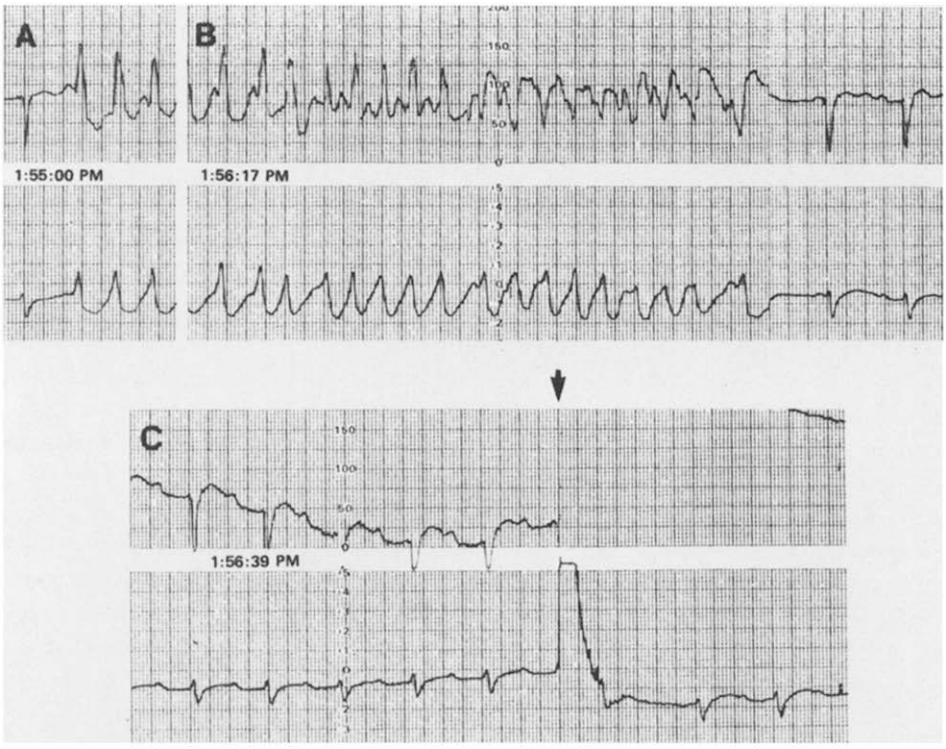

Figure 1. A, Onset of sustained monomorphous ventricular tachycardia. **B**, The tachycardia changes into a faster polymorphous rhythm and terminates spontaneously. **C**, The **arrow** shows the time of cardioverter/defibrillator discharge. See text for additional discussion.

October 11, 1984 when, after getting up in the morning, he perceived several episodes of palpitation. He reported to his local physician, who applied an ambulatory electrocardiographic monitor. A few hours later while driving his automobile, the patient experienced mild chest heaviness, soon followed by an intrathoracic shock. It was described by the patient as startling, though not painful. Because he had suffered no apparent ill effect from the shock, he was advised to remain ambulatory and no change in antiarrhythmic therapy was recommended, pending analysis of the ambulatory electrocardiogram. Several hours later while watching television, the patient experienced a second in-

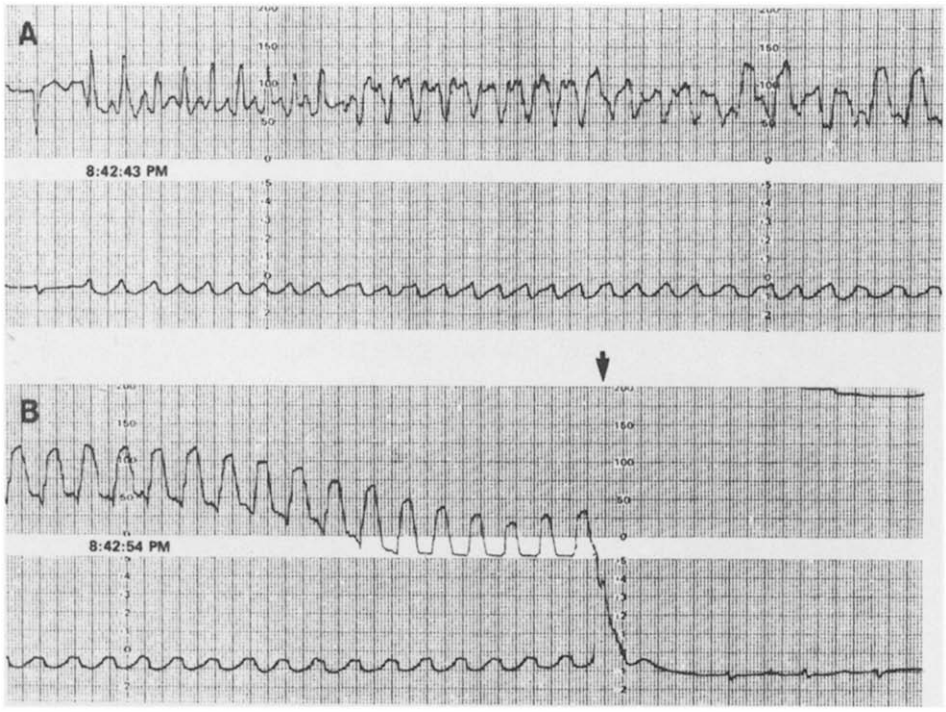

Figure 2. A, Onset of polymorphous tachycardia organizing itself into a slower monomorphous rhythm. **B**, The **arrow** shows the termination of ventricular tachycardia by cardioverter/defibrillator. See text for additional discussion.

JACC Vol. 6, No. 2
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ternal discharge, this time not preceded by any kind of subjective feeling. The 24 hour electrocardiographic monitor was removed the next day.

Ambulatory electrocardiographic data. Analysis of the 24 hour recording disclosed the occurrence of many episodes of monomorphous, nonsustained ventricular tachycardia at rates of approximately 140 beats/min. At the time of the first shock, the patient had sustained, monomorphous ventricular tachycardia at a rate of 140 beats/min (Fig. 1A) that lasted 78 seconds and then developed into a burst of self-terminating, polymorphous rhythm at an average rate of 200 beats/min for 5 seconds (Fig. 1B). Sixteen and one half seconds after the spontaneous return of sinus rhythm, the cardioverter/defibrillator delivered its first shock (Fig. 1C), activated by the last few seconds of polymorphous ventricular tachycardia. Before receiving the second shock (Fig. 2B), the patient had developed a 9 second burst of polymorphous ventricular tachycardia at an average rate of 190 beats/min (Fig. 2A) which developed spontaneously into a monomorphous, sustained rhythm at a rate of 150 beats/min and which was effectively terminated automatically by the cardioverter/defibrillator.

The patient's serum potassium level obtained on the day of the monitored events measured 3.6 mEq/liter, and the plasma amiodarone level was 1.4 μg/ml. He was advised to increase his potassium supplementation and his daily amiodarone dose from 400 to 600 mg.

Discussion

The device originally developed by Mirowski et al. (3) was created to treat ventricular fibrillation, and its first version was reliably activated by ventricular rhythms producing a sinusoidal transcardiac signal, such as ventricular fibrillation or most ventricular tachycardias at rates above 200 beats/min. It became apparent during early clinical applications, however, that a sizable proportion of patients had circulatory collapse from ventricular tachycardia sustained at rates considerably slower than 200 beats/min; such arrhythmias produced nonsinusoidal transcardiac waveforms and were consequently left untreated. This and other problems associated with a detection system based on a broad bipolar recording led to the design of a new generation of devices that incorporate in their detection algorithm both the sinusoidal configuration of the signal and a critical rate setting measured from a local ventricular bipole of the kind used in conventional cardiac pacing (4). Such refinements have been incorporated into the most recently produced version of the device, the automatic implantable cardioverter/defibrillator, markedly increasing both its sensitivity and its specificity to detect ventricular tachyarrhythmias. The trade-off for such improvements is that it can be activated by brief episodes of nonsustained ventricular tachycardia, as illustrated by our patient. In the first instance, the cardioverter/defibrillator correctly remained inactive during the first 78 seconds of slow, minimally symptomatic ventricular tachycardia, but was turned on by the last few seconds of self-terminated, rapid, polymorphous ventricular tachycardia, finally delivering a charge during sinus rhythm. In the second instance, the unit was turned on by the onset of the episode consisting of 9 seconds of rapid, polymorphous ventricular tachycardia, and was therefore committed to terminate the asymptomatic tachycardia that followed. Of note is the good subjective tolerance of both shocks by our patient, and the absence of any proarrhythmic effect of the shock delivered during sinus rhythm.

Clinical interpretation of cardioverter/defibrillator shocks. Winkle et al. (5) found it exceedingly difficult to determine on clinical grounds the precise reason why a shock has been delivered by an automatic internal cardioverter/defibrillator. We have also experienced this difficulty, particularly when shocks occur while the patient is asymptomatic. In most instances, however, readmission of the patient to our telemetry unit has allowed the observation of recurrent nonsustained ventricular tachycardia, and our efforts, as in this case, have resulted in the suppression of such runs with additional antiarrhythmic therapy. Other possibilities, such as inappropriate sinus tachycardia, paroxysmal supraventricular tachyarrhythmias and true device dysfunction, must, of course, also be considered. From a technologic point of view, correction of this adverse effect would be achieved by incorporating in the detection system a step that would confirm the need for shock within 1 or 2 seconds before its actual delivery. In our patient, the first shock would then have been prevented by the absence of ventricular tachycardia, and the second by the recognition of a tachycardia below the critical rate cut-off. The incorporation of such a feature represents, however, a technologic problem of great complexity requiring nearly beat to beat interpretation of the arrhythmia by the device's sensing system. In the case of polymorphous ventricular tachycardia or ventricular fibrillation, rate changes and signal amplitude variations may be such that an ongoing average detection over several seconds represents a much more dependable scheme than a beat to beat analysis. For now, the existing version of the automatic internal cardioverter/defibrillator remains the most reliable and successful form of treatment for selected patients with life-threatening ventricular arrhythmias.

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